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16. ABSTRACT

This manuscript is an annual literature review of disease outbreaks caused by drinking water.

Thirty-two waterborne disease outbreaks were reported to the Centers for Disease Control (CDC) and the Environmental Protection Agency in 1981.[1] The outbreaks occurred in 17 states and involved 4430 cases. This was only 64% of the number of outbreaks that were reported in 1980 and the total number of individuals involved in 1981 was less than one-quarter of that reported for the previous year. The causal agent was unknown in 44% of the 1981 outbreaks; *Giardia lamblia* was identified as the etiological agent in 28%, and chemical agents caused 16% of the outbreaks. The remaining 12% of the outbreaks were caused equally by *Shigella, Campylobacter, V. cholera 01* and rotavirus. Fifty-six percent of the outbreaks occurred in private or noncommunity water systems and the remainder were observed in community public supply systems. Most of the outbreaks (75%) were caused by the use of untreated or inadequately treated water.

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Disease outbreaks caused by drinking water

A. P. Dufour

Thirty-two waterborne disease outbreaks were reported to the Centers for Disease Control (CDC) and the Environmental

Protection Agency in 1981.[1] The outbreaks occurred in 17 states and involved 4430 cases. This was only 64% of the number of outbreaks that were reported in 1980 and the total number of individuals involved in 1981 was less than one-quarter of that reported for the previous year. The causal agent was unknown in 44% of the 1981 outbreaks; *Giardia lamblia* was identified as the etiological agent in 28%, and chemical agents caused 16% of the outbreaks. The remaining 12% of the outbreaks were caused equally by *Shigella, Campylobacter, V. cholera 01* and rotavirus. Fifty-six percent of the outbreaks occurred in private or noncommunity water systems and the remainder were observed in community public supply systems. Most of the outbreaks (75%) were caused by the use of untreated or inadequately treated water.

CAMPYLOBACTER ENTERITIS

In 1978 an outbreak of 3000 cases of diarrheal illness was reported in Bennington, Vermont.[2] The outbreak involved 19% of the population and the average duration of the illness was 4.6 days. Typical symptoms were abdominal cramps, diarrhea, and headache. The illnesses were associated with drinking water from the town water supply, which was obtained unfiltered from a nearby brook. *Campylobacter fetus* subspecies *jejuni* was cultured from 36% of 42 cases, but not from swabs of 23 individuals who served as controls. Although there was very strong epidemiological evidence linking the outbreak to the water supply, *Campylobacter* was not found in ice made at the time of the outbreak or in water samples obtained during the outbreak. Furthermore, coliform bacteria could not be found in water samples collected at the time of the epidemiological investigation or in the weeks preceding the outbreak. No further cases occurred after increased chlorination and a boil-water notice was initiated. Palmer *et al.*[3] reported an outbreak of *Campylobacter* gastroenteritis among 234 students and 23 staff members of a boarding school. The etiological agent of the outbreak, *Campylobacter jejuni,* was isolated from stool samples of both students and staff, as well as from two water samples obtained from an open-topped storage tank, which supplied unchlorinated water to the main school building. The most common serotype isolated from patients was the same as that of isolates from the water storage tank. The conclusion was drawn that the source of contamination for the water storage tank may have been fecal material from birds or bats.

YERSINIA ENTERITIS

An outbreak of gastroenteritis, which affected 87 persons, was reported from Washington state in January 1982.[4] Epidemiological evidence obtained from the outbreak investigation revealed that *Yersinia enterocolitica* was the etiologic agent, and that a strong association existed between the gastroenteritis and the ingestion of tofu, an oriental soybean curd packed in untreated spring water. Water samples from the plant water supply were positive for *Y. enterocolitica,* as were tofu samples and stool specimens from two asymptomatic employees. The installation of a water purification unit was required to eliminate the health problem.

SHIGELLOSIS

An outbreak of shigellosis affecting 928 persons residing in or near a small Canadian village was reported by Auger *et al.*[5] An epidemiological investigation indicated that the source of the infections was the village water supply and that the water carried by the municipal aquaduct was contaminated by feces containing the causal organism, *Shigella sonnei.* Single or multiple stool specimens were obtained from 220 ill persons and 59.5% of these samples were positive for *Shigella sonnei,* confirming this organism as the etiologic agent of the outbreak.

Sutmoller *et al.*[6] described a gastroenteritis outbreak which occurred in a private school in Rio de Janeiro. Examination of fecal specimens and patient sera confirmed *Shigella sonnei* and rotavirus as the microbial agents causing the outbreak. *Shigella sonnei* was isolated from 17 of 53 fecal specimens examined and a four-fold increase in antibody titer against *Shigella sonnei* was observed in 7 of 18 paired acute and convalescent sera. The source of the infections was water that was accidently pumped from a shallow well known to contain a high density of coliforms. Residual chlorine in the water supply system was zero, except in an elevated reservoir and at a kitchen tap where the value was 0.1 ppm. Foodborne transmission was eliminated because significant differences in infection rates were not observed between students who ate lunch at school and those that did not.

CHOLERA

Gunn *et al.*[7] reported 913 culture-confirmed cases of cholera caused by *Vibrio cholera El Tor.* serotype Ogawa, which occurred in Bahrain. The overall attack rate was 27 per 10 000 individuals. No common vehicle or mode of transmission was identified. However, *V. cholera* was isolated from water stored in the houses of eight cases, but not from food or tap water, implying that intrahousehold transmission may have been due to stored drinking water. It was concluded that transmission of cholera in Bahrain probably occurred through a complex interaction of contaminated food, water, and environment rather than through public water supplies.

LEGIONELLOSIS

The literature describing the occurrence of *Legionella pneumophila,* outbreaks associated with potable water, and control in water systems was reviewed by Dufour and Jakubowski.[8] Arnow *et al.*[9] described five cases of nosocomial legionellosis, all of which were exposed to aerosolized tap water from jet nebulizers or portable room humidifiers. The five patients also had received high doses of corticosteroids or adrenocorticotropic hormones. *Legionella pneumophila* was isolated from hot and cold tap water, faucet and showerhead scrapings, and from reservoirs of the aerosol generating devices. The free chlorine level in cold tap water ranged from 0.3 to 0.4 ppm and in the hot water it was less than 0.05 ppm. The initial free chlorine levels in nebulizer reservoirs was reduced from 0.4 ppm to less than 0.1 ppm in an hour and a half during operation of the nebulizer. Further cases of legionellosis were not associated with respirator devices after sterile water was substituted for tap water in the reservoirs.

Stout *et al.*[10] reported finding *Legionella pneumophila* in water samples from faucets and showerheads in a hospital just prior to an outbreak of 14 culture-confirmed cases of Legionnaires' Disease. The density of L. pneumophila ranged from 50 to 2000 colony forming units per millilitre. Although there was no direct epidemiological evidence linking water from showerheads or faucets to the cases of legionellosis, it was con-

cluded that the occurrence of the outbreak and the presence of *Legionella* in the plumbing system were probably related.

VIRAL GASTROENTERITIS

Zamotin *et al.*[11] reported an outbreak of gastroenteritis caused by rotavirus in a Russian settlement. Although a total of 173 cases were identified, it was suggested that this might be a low estimate since many individuals may not have sought assistance because of the mildness of the disease. Epidemiological evidence indicated that the central water supply system was probably the source of the infections. Rotavirus was identified as the etiological agent because it was found in the feces of 10 of 24 patients and at least a two-fold increase in antibody titer was observed in 12 of 21 paired acute and convalescent sera. The quality of the water in the distribution system was not examined.

Rotavirus also was found to be the causal agent, along with *Shigella sonnei*, of a gastroenteritis outbreak reported by Sutmoller *et al.*[6] Twenty-one percent of stool specimens examined contained rotavirus and seroconversion was noted in 22% of 18 paired acute and convalescent sera. Contaminated water from a shallow well and poor chlorination practices were indicated as the cause of the outbreak.

Kaplan *et al.*[12] reviewed records of 642 outbreaks of acute gastroenteritis to determine the proportion that were clinically and epidemiologically consistent with Norwalk-like virus infection. Forty-one percent of the outbreaks fitting the Norwalk-like pattern were waterborne. These 22 outbreaks constituted 23% of all the waterborne outbreaks reported to CDC from 1976 through 1979. The data suggested that outbreaks of acute gastroenteritis, especially waterborne outbreaks, were commonly associated with Norwalk-like viruses.

Goodman *et al.*[13] reported an outbreak of acute gastroenteritis which occurred in January 1982 in rural Georgia. A systematic survey of individuals served by the community water supply system showed that 63% had symptoms of acute gastroenteritis, whereas only 9% of those persons served by private wells and other sources had such symptoms. Twenty of 22 paired acute and convalescent sera had a four-fold increase in antibody titer to Norwalk virus. Fecal coliforms were detected in spring water which served as one source for the community water system. A heavy rainfall that preceded the outbreak may have contributed to the contamination of the system.

Another outbreak of gastroenteritis in Georgia caused by Norwalk virus that occurred in August of 1980 was described by Kaplan *et al.*[14] An association between gastrointestinal illness and consumption of drinking water was shown for community residents, students, and school staff. The attack rates increased significantly with proximity to a textile plant where two known cross-connections existed between the industrial water system and the community water system. Sixty-three percent of 19 patient serum pairs showed a four-fold rise in antibody titer to Norwalk virus, confirming the etiologic agent of the outbreak.

Wilson *et al.*[15] reported an outbreak of gastroenteritis at a childrens' summer recreational camp in northeastern Pennsylvania. The illness was associated with the consumption of drinking water. Although stool specimens examined by electron microscopy were negative for viruses, paired sera from three acutely ill individuals showed a four-fold rise in titer to Norwalk agent. Well and kitchen tap water samples contained total coliforms and fecal streptococci.

HEPATITIS A

A waterborne outbreak of gastroenteritis and hepatitis, which occurred in a small Texas community, was reported by Hejkal *et al.*[16] Thirty-six hepatitis A cases were observed over a one-month period in a community where the expected incidence was 0 to two cases per month. An epidemiological survey indicated that the outbreak was associated with water from the central-city wells. Bacteriological data were not available for the period prior to the outbreak, but at about the time the outbreak peaked, 125 water samples from the distribution system were examined and all were negative for coliforms. However, large numbers of fecal coliforms were present in the city wells following a second peak of gastroenteritis and human enteric viruses were isolated from the potable water in spite of a chlorine residual of 0.8 ppm. It was concluded that this level of chlorine was sufficient to kill coliforms but was not sufficient to kill viruses.

Linglof *et al.*[17] described a waterborne hepatitis A epidemic in Uppland, Sweden. The outbreak involved about 30 persons whose illnesses were confirmed serologically. Epidemiological evidence implicated drinking water as the source of the infections. A conclusion of this report was that the risk of hepatitis applied mainly to small, insufficiently controlled, private water supplies.

TOXOPLASMOSIS

An outbreak of acute toxoplasmosis among soldiers training in the Panama Canal Area was reported by Benenson *et al.*[18] Two weeks subsequent to the training exercise, after their return to North Carolina, 39 of 98 soldiers in one company came down with a febrile illness and three of the individuals required hospitalization. *Toxoplasma gondii* was confirmed seriologically as the etiologic agent of the outbreak. The source of the infectious agent was two jungle streams from which water was collected for drinking purposes. Only ingestion of jungle creek water, probably contaminated by oocysts containing feces from infected jungle cats, accounted for the distribution of infection that was observed among the soldiers.

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